(19)日本国特許庁(JP)

(12) 公開特許公報(A)

(11)特許出願公開番号

特開平5-98147

(43)公開日 平成5年(1993)4月20日

(51)Int.Cl.⁵

識別記号 LQP 庁内整理番号

9167 - 4 J

FΙ

技術表示箇所

C08L 71/12

// (C 0 8 L 71/12

9: 00

25: 04

53:00)

審査請求 未請求 請求項の数1(全 8 頁)

(21)出願番号

特顯平3-257515

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(22)出願日 平成3年(1991)10月4日

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(54) 【発明の名称 】 変成ポリフエニレンエーテル系樹脂組成物

(57) 【要約】

【構成】 (A) 不飽和カルボン酸またはその誘導体で変性処理された変性ポリフェニレンエーテル系樹脂、

(B) 1, 4ーシス結合を50%以上有しかつ1, 2ービニル結合が10%以下であるハイシス共役ジエン系ゴムを含む耐衝撃性スチレン系樹脂、(C) スチレン系樹脂、(D) 不飽和カルボン酸またはその誘導体で変性処理されたスチレン系ポリマーブロックとオレフィン系エラストマーブロックとより成る変性ブロック共重合体樹脂、の(A) 成分、(B) 成分、(C) 成分、(D) 成分より成るポリフェニレンエーテル系樹脂組成物。

【効果】 本発明の組成物は、優れた耐衝撃性、熱安定性と、優れた成形加工性、塗装密着性等の二次加工性を併せ持つ大型成形用材料に関する。

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Inhibition of the Cardiac p38-MAPK Pathway by SB203580 Delays Ischemic Cell Death

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Summary: We report that SB203580 (SB), a specific inhibitor of p38-MAPK, protects pig myocardium against ischemic injury in an in vivo model. SB was applied by local infusion into the subsequently ischemic myocardium for 60 min before a 60-min period of coronary occlusion followed by 60-min reperfusion (index ischemia). Infarct size was reduced from a control value of $69.3 \pm 2.7\%$ to $36.8 \pm 3.7\%$. When SB was infused systemically for 10 min before index ischemia, infarct size was reduced to $36.1 \pm 5.6\%$. We measured the content of phosphorylated p38-MAPK after systemic infusion of SB and Krebs-Henseleit buffer (KHB; negative control) and during the subsequent ischemic period using an antibody that reacts specifically with dual-phosphorylated p38-MAPK (Thr180/ Tyr182). Ischemia with and without SB significantly increased phospho-p38-MAPK, with a maximum reached at 20 min but was less at 30 and 45 min under the influence of the inhibitor. The systemic infusion of SB for 10 min before index ischemia did not significantly change the p38-MAPK activities (compared with vehicle, studied by in-gel phosphorylation) ≤20 min

of ischemia, but activities were reduced at 30 and 45 min. Measurements of p38-MAPK activities in situations in which SB was present during in-gel phosphorylation showed significant inhibition of p38-MAPK activities. The systemic infusion of SB significantly inhibited the ischemia-induced phosphorylation of nuclear activating transcription factor 2 (ATF-2). Using a specific ATF-2 antibody, we did not observe significant changes in ATF-2 abundance when nuclear fractions from untreated, KHB-, and SB-treated tissues were compared. We investigated also the effect of local and systemic infusion of SB on the cardioprotection induced by ischemic preconditioning (IP). The infusions (local or systemic) of SB before and during the IP protocol did not influence the infarct size reduction mediated by IP. The observed protection of the myocardium against ischemic damage by SB points to the negative role of the p38-MAPK pathway during ischemia. Key Words: SB203580-Protein kinases-p38-MAPK-Ischemia/reperfusion-Pig.

Stressful stimuli applied as brief pulse trains condition most tissues so that they become more tolerant against longer lasting stresses, significantly delaying the onset of irreversible damage (1). The molecular mechanism of this increase in stress tolerance, especially that toward ischemia, is not entirely clear, but mitogen activated protein kinases (MAPKs), which are involved in the signal-transduction pathways, may play a role. In previous reports we have shown that brief ischemic pulses lead to changes in the expression of the cardiac protooncogenes that may participate in the adaptive response (2,3). Since the protooncogene-based transcription factors are activated by membrane and cytoplasmic signaling cascades, we studied the involvement of three MAPK pathways

(extracellular signal-regulated kinases, ERKs; stress-activated/c-Jun N-terminal kinases, SAPK/JNKs; and p38-MAPK) and found that they react to brief ischemia and reperfusion in a very specific way: the ERKs moderately increased activity during brief ischemia but markedly during reperfusion, the SAPK/JNKs become active only during reperfusion, and the p38-MAPK was activated only during ischemia and deactivated during the following reperfusion and subsequent period of ischemia (4). MAPK pathways can also be influenced by pharmacologic agents that specifically influence the activity of members of the MAPKs, particularly those of the stressactivated protein kinases (SAPK/JNKs, p38-MAPK) (5,6). A common feature of all MAPKs is their

Received July 28, 1999; revision accepted November 15, 1999.
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ability to phosphorylate the transactivation domains of numerous transcription factors and thus modulate transcriptional activity. However, it cannot be excluded that MAPK activation also has effectors outside the nucleus. The fact that repeated brief occlusion, which can amplify the conditioning effect of the first occlusion, induced an attenuation of p38-MAPK activity suggested that p38-MAPK activation may cause premature ischemic cell death. We hypothesized that SB203580 (SB), an inhibitor of p38-MAPK, may be able to protect the heart against the consequences of prolonged ischemia. This hypothesis was tested in an in vivo model by two different application methods of SB: intramyocardial and intravenous infusion.

MATERIALS AND METHODS

The experimental protocol described in this study was approved by the Bioethical Committee of the District of Darmstadt, Germany. Furthermore, all animals in this study were handled in accordance with the guiding principles in care and use of animals as approved by the American Physiology Society, and the investigation conformed with the Guide for Care and Use of Laboratory Animals published by the U.S. National Institutes of Health.

Chemicals

Azaperone, metomidate, and piritramide were purchased from Janssen Pharmaceutica (Meckenheim, Germany). SB, protein kinase inhibitor (PKI), α-chloralose, triphenyl tetrazolium chloride (TTC), and other biochemicals were from Sigma (Deisenhofen, Germany). The fluorescent zinc-cadmium sulfide microspheres (diameter, 2-15 µm) were purchased from Duke Scientific Corp (AC Leusden, Netherlands). The polyclonal antibody against p38-MAPK was from Santa Cruz Biotechnology (Heidelberg, Germany). Phospho-p38-MAPK, phospho-SAPK/JNK, ATF-2, and phospho-ATF-2 antibodies were from New England Biolabs (Schwalbach/Taunus, Germany). Nitrocellulose membranes, rainbow molecular mass markers, the horseradish peroxidase-linked goat anti-rabbit immunoglobulin, the enhanced chemiluminescence (ECL) reagents, autoradiography films, and $[\tau^{-32}P]$ -ATP were from Amersham (Pharmacia Biotech, Europe GmbH, Freiburg, Germany). Recombinant MAPKAPK-2 (residues 46-400 encompassing the catalytic domain) was expressed in Escherichia coli as glutathione-S-transferase fusion protein (clone provided by C. J. Marshall, a kind gift from P. H. Sugden) and was purified by glutathione-Sepharose (Pharmacia) chromatography.

Animal preparation

Male castrated German landrace-type domestic pigs $(32.6 \pm 2.3 \text{ kg})$ were premedicated with azaperone (2 mg/kg) of body weight, i.m.) and 2 mg/kg BW piritramide, s.c., 30 min before the initiation of anesthesia with 10 mg/kg BW metomidate. After tracheal intubation, a bolus of α -chloralose (25 mg/kg) was given intravenously. Anesthesia was maintained by a continuous intravenous infusion of α -chloralose (25 mg/kg/h). The animals were ventilated artificially with a pressure-controlled respirator (Stephan Respirator ABV, F. Stephan GmbH, Gackenbach, Germany) with room air enriched with 2 L/min of oxygen. Arterial blood gases were analyzed frequently to guide

adjustment of the respirator settings. Additional doses of piritramide (10 mg) were given i.v. every 60 min. Both internal jugular veins were cannulated with polyethylene tubes for administration of saline, piritramide, and α -chloralose. Arterial sheath catheters (7F) were inserted into both carotid arteries. To measure arterial blood pressure, the left sheath was advanced into the aortic arch and connected with a Statham transducer (P23XL; Statham, San Juan, Puerto Rico). After a midsternal thoracotomy, the heart was suspended in a pericardial cradle. Arterial pressure, heart rate, and the ECG were continuously monitored and recorded on the hard disk of a MacLab computer. A loose reversible ligature was placed halfway around the left anterior descending artery (LAD), and was subsequently tightened to occlude the vessels. In pigs subjected to intramyocardial microinfusion, eight 26-gauge needles connected by tubing with a peristaltic pump (Miniplus; Gilson, Villiers-le-Bel, France) were placed in pairs along the LAD into the myocardium perpendicular to the epicardial surface. After preparation, a stabilization period of 30 min was allowed and the experimental protocols were started. The p38-MAPK inhibitor, SB, was dissolved in DMSO and finally diluted in Krebs-Henseleit buffer (KHB; final concentration of DMSO was 0.1%). For this reason, the infusion of KHB with DMSO served as a negative control (KHB).

Experimental groups

This study consisted of eight experimental groups (Fig. 1). Group I was subjected to 60 min of occlusion and 60 min of reperfusion (control group 1). In group II, SB (40 nM) or KHB (with 0.1% DMSO) was administered by local infusion for 60 min before the index ischemia of 60 min and the following reperfusion period of 60 min. In group III, SB (5 mg/animal) or KHB was applied by systemic infusion for 10 min before the index ischemia (60 min occlusion and 60 min reperfusion periods). In group IV, the animals were subjected to 40 min of occlusion followed by 60 min of reperfusion (control group 2). In group V, the animals were subjected to the preconditioning protocol (two cycles of 10-min ischemia and 10-min reperfusion) followed by a period of 40-min index ischemia and 60 min of reperfusion. In group VI, SB (40 nM) or KHB was administered by local microinfusion for 15 min before the brief occlusions/reperfusions and during reperfusion periods of the preconditioning protocol. This was followed by 40 min of ischemia and 60 min of reperfusion. In group VII, SB (5 mg/ animal) or KHB was applied by intravenous infusion for 15 min before the brief occlusions/reperfusions and during reperfusion periods of the preconditioning protocol. This was followed by 40 min of index ischemia and 60 min of reperfusion. In group VIII, SB (5 mg/animal) or KHB was applied by intravenous infusion for 10 min before the index ischemia of 60 min, and left ventricular biopsies for in vitro assays were obtained at the end of SB and KHB infusion and at 5, 10, 20, 30, 45, and 60 min of the following index ischemia. Drill biopsies were taken from control tissue, KHB-, and SB-treated tissue (Fig. 1). Biopsies weighed -80 mg and were -4 mm long (i.e., they reached from epi- to midmyocardium).

Determination of infarct size

At 45 min into the last reperfusion period, 1 g of fluorescein dissolved in 10 ml Ringer's solution was injected into the right ventricle. This stained the entire myocardium and detected non-reperfused tissue. Hearts with traces of nonreperfused myocardium were excluded from analysis. At the end of the experi-

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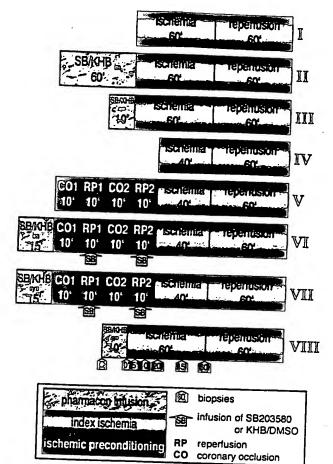


FIG. 1. This study consisted of eight experimental groups. Group I was subjected to 60 min of occlusion and 60 min of reperfusion (control group 1). In group II, SB203580 (40 nM) or KHB (with 0.1% DMSO, negative control) were administered by local intramyocardial infusion (i.m.; infusion rate, 20 µl/min) for 60 min before the index ischemia of 60 min and the following reperfusion period of 60 min. In group III, SB203580 (5 mg/animal) or KHB was applied by systemic infusion (sys) for 10 min before the index ischemia of 60 min and the following reperfusion period of 60 min. In group IV, the animals were subjected to 40 min of occlusion followed by 60 min of reperfusion (control group 2). In group V, the animals were subjected to a preconditioning protocol of two cycles of 10-min ischemia and 10-min reperfusion followed by the index ischemia of 40-min occlusion and a reperfusion period of 60 min. In group VI, SB203580 (40 nM) or KHB was administered by local microinfusion for 15 min before preconditioning and during the reperfusion phase of the preconditioning protocol. This was followed by index ischemia of 40-min occlusion and a reperfusion period of 60 min. In group VII, SB203580 (5 mg/animal) or KHB was applied by systemic infusion for 15 min before preconditioning and during the reperfusion phases. This was followed by the index ischemia of 40-min occlusion and the reperfusion period of 60 min. In group VIII, SB203580 (5 mg/ animal) or KHB was systematically infused for 10 min before index ischemia of 60 min, and left ventricular biopsies for in vitro assays were obtained at the end of SB and KHB infusion and at various intervals of index ischemia.

mental protocol, the LAD and the aorta were occluded, clamped, and 500 mg of zinc cadmium fluorescent microspheres in 10 ml of Ringer's solution were injected into the ascending aorta. Shortly thereafter, the animals were injected

with an intravenous bolus of 20% potassium chloride to arrest the heart. After excision, both atria and the right ventricle were removed. The left ventricle was cut into slices along the microinfusion needle pairs perpendicular to the LAD. Heart slices were weighed and incubated at 37°C in 1% triphenyltetrazolium chloride (TTC) in PBS, pH 7.0, for 20 min. Myocardium at risk of infarction was identified as the nonfluorescent (by microspheres) area by UV light (366 nm). The infarcted area was demarcated by the absence of the characteristic red TTC stain. The slices were photographed by double exposure with UV and artificial daylight, and the pictures were used for further planimetric evaluation. Planimetry of the infarct areas was performed on the basal aspect of the apex, the apical and basal sides of the following four consecutive myocardial slices, and on the apical aspect of the basal section of the left ventricle. We expressed infarct size (IS) as the infarct area (IA) relative to the risk area (RA). Infarct sizes were then averaged per group and depicted graphically (Figs. 3 and 5).

Preparation of soluble and nuclear fractions

The biopsies for the kinase assays were suspended in icecold buffer containing in mM: 20 Tris-HCl, 250 sucrose, 1.0 EDTA, 1.0 EGTA, 1.0 dithiothreitol (DTT), 0.1 sodium orthovanadate, 10 NaF, and 0.5 PMSF, pH 7.4, (buffer A), and were homogenized with a Teflon-glass homogenizer. The homogenates were centrifuged at 14,000 g for 30 min at 4°C. The supernatants represented the soluble (cytosolic) fractions. The pellets were resuspended in buffer B containing in mM: 20 Tris-HCl, 1,000 sucrose, 1.0 EDTA, 1.0 EGTA, 1.0 DTT, 0.1 sodium orthovanadate, 10 NaF, 10 KCl, and 0.1 PMSF (pH 7.4), and were centrifuged for 30 min at 10,000 g (4°C). The resulting pellets were resuspended in buffer C containing 10% glycerol, 20 mM Tris-HCl, 400 mM KCl, 1.0 mM EGTA, 1.0 mM DTT, 0.1 mM sodium orthovanadate, 10 mM NaF, 0.5 mM PMSF, and 0.1% Triton X-100, and sonicated and used for the detection of transcription factor ATF-2. For the preparation of electrophoretic probes, Laemmli sample buffer was added and the proteins were denatured by heating. The denatured probes were applied to sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE), and used for MAPK assays by in-gel kinase assays and for Western blot analysis.

Measurement of p38-MAPK activities by in-gel phosphorylation

Equivalent amounts of proteins were separated on 10% SDSpolyacrylamide gels containing 0.5 mg/ml of GST-MAPKAPK-2₄₆₋₄₀₀. After electrophoresis, the gels were washed for 1 h with 20% (vol/vol) 2-propanol in 50 mM Tris-HCl (pH 8.0), followed by 1 h with 5 mM 2-mercaptoethanol in 50 mM Tris-HCl, pH 8.0. The in-gel proteins were denatured by incubation for 2 h with 50 mM Tris-HCl, pH 8.0, containing 6 M guanidine-HCl. Renaturation was achieved by incubation with 50 mM Tris-HCl, pH 8.0, containing 0.1% (vol/vol) Nonidet P-40 and 5 mM 2-mercaptoethanol for 16 h. After preincubation of gels in 40 mM HEPES (pH 8.0) containing 2 mM DTT and 10 mM magnesium chloride, the in-gel phosphorylation of substrates was performed in 40 mM HEPES (pH 8.0), 0.5 mM EGTA, 10 mM magnesium chloride, 1.0 µM protein kinase A inhibitory peptide, and 25 μM [τ^{-32} P]-ATP (5 μ Ci/ ml) at 25°C for 4 h. In some experiments, the sample preparation, incubation of gels, and phosphorylation were performed in the presence of 50 nM SB. After extensive washing in 5% (wt/vol) trichloroacetic acid containing 2% (wt/vol) sodium pyrophosphate, the gels were dried, and quantitative analysis was

ブロック共重合体の併用が可能であり、併用できる量は カルボン酸基またはその誘導体基を含有する分子単位の 量が変性及び未変性のブロック共重合体の合計に対し、 O. 05重量部以上であり、且つ未変性ブロック共重合 体の量が変性ブロック共重合体の量を上回らないことが 好ましい。

【〇〇25】本発明において、(A) 成分の変性ポリフェニレンエーテル系樹脂の含有量は10~90重量部の範囲が好ましい。含有量が10重量部未満の場合には、変性ポリフェニレンエーテル系樹脂による改良効果が十分発揮されないため好ましくなく、90重量部を超える場合、成形加工性あるいは耐衝撃性スチレン系樹脂を添加することが出来なくなるため好ましくない。

【〇〇26】(C)成分は、主として組成物の加工性及び熱変形温度の調節のために用いられる。該成分の量が80重量部を超えると、ポリフェニレンエーテル系樹脂の持つ特性を大きく損なうため好ましくない。(D)成分は、耐衝撃性及び耐油性を更に改良するのに有用である。しかし、該成分の量が20重量部を超えると、剛性の極端な低下が生ずるため好ましくない。

【〇〇27】本発明の組成物には他の添加剤、例えば、可塑剤、安定剤、紫外線吸収剤、難燃剤、着色剤、離型剤及びガラス繊維、炭素繊維等の繊維状補強剤、更にはガラスビーズ、炭酸カルシュウム、タルク等の充填剤を添加することができる。安定剤としては、亜リン酸エステル類、ヒンダードフェノール類、アルカノールアミン類、酸アミド類、ジチオカルバミン酸金属塩類、無機硫化物、金属酸化物類の中から単独でまたは組み合わせて使用することができる。

【OO28】難燃剤としては、芳香族リン酸エステル、赤燐、芳香族ハロゲン化合物、三酸化アンチモン等が特に有効である。本発明を構成する各成分を混合する方法はいかなる方法でもよい。例えば、押出機、加熱ロール、バンバリーミキサー、ニーダー等を使用することが出来る。

[0029]

【実施例】以下に実施例によって本発明を具体的に示すが、本発明は以下の例に限定されるものではない。以下、%及び部は、夫々重量%及び重量部を表す。なお、以下の実施例及び比較例において用いた耐衝撃性スチレン系樹脂は次に述べる製造方法によって調製したものである。また、実施例及び比較例で用いた耐衝撃性スチレン系樹脂とこれを調製するに際して用いた共役ジエン系ゴム及びハイシス共役ジエン系ゴムは表2に示す通りのものである。

【OO30】製造例1;耐衝撃性スチレン系樹脂の製造 occlusion followed by reperfusion of 60 min (control group 1).

ス結合 3%であり、5%スチレン溶液粘度が65センチポイズであるハイシスポリブタジエン10部をスチレンりの部とエチルベンゼン8部に溶解し、更にスチレンに対して0.05部のベンゾイルパーオキサイドと0.01部のαーメチルスチレン2量体を添加し、80℃で4時間、110℃で4時間、150℃で4時間、150℃で4時間、150℃で4時間、攪拌に重合を行った。更に230℃前後で30分間加熱処理を行い、その後、未反応スチレン及びエチルベンゼンのの強力を行い、耐衝撃性スチレン系樹脂を得た。得られた耐衝撃性スチレン系樹脂やのポリブタジエンの合有状にでの部分水添ポリブタジエンの平均粒子径は2.7μであった。

【 O O 3 1 】製造例2:変性ポリフェニレンエーテル樹脂の調製 { (A) 成分} 、

極限粘度 O. 50 (クロロホルム中、30℃にて測定)のポリ(2.6ージメチルー1,4ーフェニレン)エーテル(PPE)100部に対して、ポリスチレン50部、無水マレイン酸2部、パーブチルDO.5部を均一に混合した後、押出機を用い、窒素雰囲気下で300℃にて溶融混練しマレイン化反応を行い、変性ポリフェニレンエーテル樹脂混合物を得た。ナトリウムメチラートによる滴定によって求めたポリフェニレンエーテル樹脂100部に対する無水マレイン酸の付加量は0.8部であった。

【OO32】実施例において、組成物中の成分に対する 無水マレイン酸の付加量は次のように測定した。組成物 を38℃にてジクロロメタンに溶解した後これを濾過 し、次いで、溶液部分を-5℃にて24時間放置し、変 性ポリフェニレンエーテル系樹脂を析出させ、これを濾 過し、該樹脂を分離した。次に、濾液をメタノールに滴 下し、ポリスチレンを再沈澱させ濾過し、これを分離し た。各成分に対する無水マレイン酸の付加量はナトリウ ムメチラートによる滴定によって測定した。

【0033】尚、ポリスチレンに対する無水マレイン酸の付加は認められなかった。

製造例3;変性ブロック共重合体樹脂の調製 ((D)成分)、

スチレン系ポリマーブロックとオレフィン系エラストマーブロックとより成るブロック共重合体100部に対して、無水マレイン酸2部、パーブチルロ(日本油脂社製)0.5部を均一に混合した後、押出機を用い260℃にて溶融混練しマレイン化反応を行い、変性ブロック共重合体混合物を得た。ナトリウムメチラートによる滴定によって求めた変性ブロック共重合体樹脂のブロック共重合体に対する無水マレイン酸の付加量は1.0~

極限粘度 0.50 (クロロホルム中、30℃にて測定)のポリ(2、6ージメチルー1、4ーフェニレン)エーテル(PPE)50部に対して、スチレン系ポリマーブロックとオレフィン系エラストマーブロックとよりなるブロック共重合体 17部、ポリスチレン33部、無水マレイン酸 0.5部、パーブチル D 0.25部を均一に混合した後、押出機を用い、窒素雰囲気下で300℃にて溶融混練し、マレイン化反応を行い、変性ポリフェニレンエーテル樹脂混合物を得た。ナトリウムメチラーによる滴定によって求めたポリフェニレンエーテル樹脂 100 部に対する無水マレイン酸の付加量は 0.2 部であった。

【0035】実施例において、組成物中の成分に対する無水マレイン酸の付加量は次のように測定した。組成物を38℃にてジクロロメタンに溶解した後これを濾過し、次いで、溶液部分を-5℃にて24時間放置し、変性ポリフェニレンエーテル樹脂を析出させ、これを濾過し、該樹脂を分離した。次に、濾液をメタノールに滴下しポリスチレンを再沈澱させ濾過し、これを分離した。各成分に対する無水マレイン酸の付加量はナトリウムメチラートによる滴定によって測定した。

【〇〇36】尚、ポリスチレンに対する無水マレイン酸の付加は認められなかった。変性ブロック共重合体樹脂のブロック共重合体に対する無水マレイン酸の付加量は〇.8~1.2部であった。次に樹脂組成物の物性測定評価方法について説明する。

(1) 溶融指数

JIS K 7210に準拠

温度:250℃, 荷重:10Kg

射出成形機(東芝機械株式会社製、IS80C、シリンダー温度280℃または290℃)で試験片を作成し、以下の(2)~(5)を測定した。

【0037】(2)熱変形温度

ASTM D648, 荷重:18.6Kg/cm²

(3) アイゾット衝撃強さ

ASTM D256, ノッチ有り

温度:23℃

(4) 成形安定性

射出成形温度280℃(または290℃)で成形した成形品のアイゾット衝撃強さに対する310℃(または320℃)で10分間(または15分間)シリンダー内で滞留させた後、成形した成形品のアイゾット衝撃強さの保持率から判定した。

【0038】射出成形機(東芝機械株式会社製、IS8 0C、シリンダー温度290℃)で75×75×3mm 試験片を作成し、以下の(7)~(8)を測定した。

(5) 塗装密着

市販のウレタン系塗料で塗装後、碁盤目試験を実施し、 全碁盤目 (100個) 中の剥離した碁盤目の数により下 記ランクをつけた。

[0039]

②: 剥離数が 0〇: 剥離数が 1~ 10Δ: 剥離数が 11~ 20×: 剥離数が 21~100

[0040]

【実施例1】製造例2で作成した変性ポリフェニレンエーテル系樹脂35部、表1のNo.1の耐衝撃性ポリスチレン(以下HIPSと略称する)54部、製造例3で作成した変性ブロック共重合体樹脂(無水マレイン酸付加量1.0部)11部及びスミライザーBHT(住友化学製のヒンダードフェノール)1部とを、押出機を用いて280℃の温度にて溶融混練して樹脂組成物を得た。該樹脂組成物の物性試験結果を表2に示す。

[0041]

【実施例2】製造例2で作成した変性ポリフェニレンエーテル系樹脂7部、極限粘度 0.50 (クロロホルム中、30℃にて測定)のポリ(2.6ージメチルー1.4ーフェニレン)エーテル(以下PPEと略称する)28部、表1のNo.1の耐衝撃性ポリスチレン(以下HIPSと略称する)54部、製造例3で作成した変性ブロック共重合体樹脂(無水マレイン酸付加量1.0部)11部及びスミライザーBHT(住友化学製のヒンダードフェノール)1部とを、押出機を用いて280℃の温度にて溶融混練して樹脂組成物を得た。該樹脂組成物の物性試験結果を表2に示す。

[0042]

【実施例3】製造例4で作成したポリフェニレンエーテルとブロック共重合体の同時変性体46部、表1のNo.1の耐衝撃性ポリスチレン(以下HIPSと略称する)54部及びスミライザーBHT(住友化学製のヒンダードフェノール)1部とを、押出機を用いて280℃の温度にて溶融混練して樹脂組成物を得た。該樹脂組成物の物性試験結果を表2に示す。

[0043]

【比較例 1 】極限粘度 0. 50 (クロロホルム中、30 ℃にて測定)のポリ(2,6ージメチルー1,4ーフェニレン)エーテル(以下PPEと略称する)を実施例 1の変性ポリフェニレンエーテル系樹脂の代わりに用いた以外は実施例 1 と同様の操作を行った。結果を表 2 に示す。

[0044]

【比較例2】表1のNo. 1のHIPSに替えて、表1のNo. 2のHIPSを用いた以外は比較例1を繰り返して樹脂組成物を得た。結果を表2に示す。

[0045]

【実施例4】製造例2で作成した変性ポリフェニレンエーテル系樹脂45部、表1のNo. 1のHIPS55部及びスミライザーBHT(住友化学製のヒンダードフェノール)1部とを、押出機を用いて280℃の温度にて

溶融混練して樹脂組成物を得た。該樹脂組成物の物性試 験結果を表3に示す。

[0046]

【実施例5】製造例2で作成した変性ポリフェニレンエーテル系樹脂9部、極限粘度 O. 50 (クロロホルム中、30℃にて測定)のポリ(2、6ージメチルー1、4ーフェニレン)エーテル(以下PPEと略称する)36部、表1のNo. 1のH1PS55部及びスミライザーBHT(住友化学製のヒンダードフェノール)1部とを、押出機を用いて280℃の温度にて溶融混練して樹脂組成物を得た。該樹脂組成物の物性試験結果を表3に示す。

[0047]

【比較例3】実施例3で使用した変性ポリフェニレンエーテル系樹脂の代わりに極限粘度 0. 5 0 (クロロホルム中、30℃にて測定)のPPEを用いた以外は実施例3を繰り返して樹脂組成物を得た。該樹脂組成物の物性試験結果を表3に示す。

[0048]

【比較例4】表1のNo. 1のHIPSに替えて、表1のNo. 2のHIPSを用いた以外は比較例3を繰り返して樹脂組成物を得た。結果を表3に示す。

[0049]

【表1】

耐衝撃性スチレン系樹脂 No.			1	2
共役ジェン系ゴム健			ポリプ♭ シ゚エン	ポリプタ シ゚エン
	5 % S V		65	65
	1,2-	:=ル (%)	3	13
7 4	1,4-	シス (%)	94	3 4
	极造	トランス(%)	3	53
耐衝擊性	スチレン化	公合物	スチレン	スチレン
スチレン	1 A 1	i (%)	1 1	1 1
系樹脂	平均粒子包	ξ (μ)	2.7	2.8

[0050]

【表2】

		実施例 1	実施例 2	実施例 3	比較例	比較例 2
溶触指数	(g/10min.)	1 3	8	1 2	6	6
熱変形温度	(℃)	115	115	115	1 1 5	115
アイソット 衝撃強	さ (kgcm/cm)	27	29	3 0	28	23
成形安定性	(%)	7 8	78	79	78	7 2
绘装密 验性		0	0	0	Δ	Δ

射出成形温度290℃、成形安定性は320℃で15分間滞留。

[0051]

【表3】

	実施例 4	実施例 5	比較例 3	比較例 4				
浴融指数 (g/10min.	1 1	7	5	5				
熱変形温度 (℃)	120	120	120	120				
アイソット 衝撃強さ (kgcm/cm)	1 3	1 5	1 4	12				
成形安定性 (%)	6 0	60	60	50				
捡装密 脊性	· ©	0	Δ	Δ				

射出成形温度280℃、成形安定性は310℃で10分間滞留。

[0052]

【発明の効果】本発明の組成物は溶融加工工程または熱 暴露などの加熱によって物性低下が起こらず、しかも従 来の樹脂組成物に比べ耐衝撃性、塗装密着性等のバランスが優れ、特に大型成形に適した産業上有用なものである。